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Exercise-associated hyponatraemia: facts and myths

In an elaborate manuscript, Noakes and Speedy argue that exercise-associated hyponatraemia (EAH) is due to overhydration and that the usual sodium deficit incurred during exercise plays an insignificant role.[1 2] Furthermore, these researchers claim that the reluctance to accept their evidence may be because it conflicts with the prevalent message of the sports drink industry. This approach is, however, oversimplification of the situation and at best singlesided. Noakes and Speedy in actuality prove the obvious. Overhydration will dilute body fluid, and thus plasma sodium concentration, as the primary extracellular electrolyte, will consequently be reduced. However, because hyponatraemia can be observed in the presence of a normal, depleted or expanded plasma volume, the issue of hyponatraemia should be approached from a broader perspective. We emphasise that the inability to replace cumulative sodium losses in sweat during long physical exertions is not less important as the background for EAH, whereas hyperhydration is only a marginal, although vastly reported, condition associated with EAH.

Body fluid homoeostasis is a primary variable, which is important in the stability of the ''inner environment''. Therefore, total body water and the proportion of extracellular to intracellular volumes should be maintained very delicately. Dehydration during exercise results in graded physiological responses, including a decrease in cardiac output despite an increase in heart rate, an increase in core temperature and a decrease in peripheral blood flow, which ultimately affects performance. Eichna *et al*[3] described the ability of dehydrated participants' to perform in the heat as: ''total incapacitation … acclimatised subjects who had performed a given task easily … reduced to apathetic, listless, plodding men straining to finish the same task''. Fluid replacement during exercise contributes to the maintenance of plasma volume, aiding thermal and cardiovascular homoeostasis and thus reversing all these adverse effects.[4 5] Therefore, dehydration should not be underestimated: it will not only adversely influence performance but will also, when severe, threaten life. Sport specialists who advocate fluid replacement ad libitum actually disregard the ill effects of dehydration. However, optimal rehydration should not be withheld only out of fear for overhydration.

To illustrate the point that overhydration is not the sole reason for EAH, we analyse the following examples: Hew-Butler *et al*[6] show that following the South African Ironman triathlon (lasting 12:20 h), serum [Na^+] was maintained at 140.9 mEq/l (baseline levels were 140.5 mEq/l). This was achieved by drinking ad libitum, thus attaining a dehydration level of 3.9%. If the athletes would have drunk water according to sweat loss, with no overhydration at all, final serum [Na^+] would have been in the magnitude of 134.7 mEq/l (assuming a sweat rate of 450 ml/h and a sweat sodium concentration of 50 mEq/l). Similarly, Montain *et al*[7] show that serum [Na^+] of a 70 kg male athlete after a 9 h, 90 km ultramarathon foot race will be 130.2 mEq/l if [Na^+] in sweat is 50 mEq/l and if he drinks water to maintain euhydration. By recalculating the data, assuming that the athlete would have drunk ad libitum, incurring a mild state of dehydration at an estimate level of 4%, serum [Na^+] after the race will be higher, 135.5 mEq/l. These two examples well illustrate that during long physical exertions, overhydration is not the reason for lowering serum [Na^+] but rather the cumulative loss of sodium in sweat that is not adequately replenished. This opinion is substantiated in the following observation by Y Epstein. Two groups of soldiers participated in a 50 km (10 hr) intense foot march. Group A drank water at a rate that compensated sweat loss (1000 ml/h). Group B drank the same amount of water but were also served with snacks and sandwiches, which they consumed ad libitum (average total sodium consumption = 222 mEq). At the end of the exercise, the mean (SD) serum [Na^+] of participants in group A was 134 (1) mEq/l, whereas in group B it was138 (1) mEq/l (pre-exercise [Na^+] = 142 mEq/l). The consumption of sodium by participants in group B has attenuated the drop in plasma [Na^+]. This finding is corroborated by Vrijens and Rehrer, who showed that sodium consumption can slow down the drop in plasma [Na^+] after long physical exercise.[8] The small reduction in serum [Na^+] in group B is due to the less than desirable sodium intake. Assuming a sweat [Na^+] of 40–50 mEq/l, these soldiers replaced only about 50% of the amount of sodium loss.

Is salt replacement needed all the time? The answer is probably ''no''. Sweat is a hypotonic fluid and the reported [Na^+] in sweat is in the range of 20–80 mEq/l.[9] In general, [Na^+] in the sweat of fit acclimated subjects is in the range of 20–40 mEq/l, whereas in unfit subjects it is nearly twofold higher.[10] Thus, when water intake is inadequate to compensate sweat loss and dehydration ensues, the blood becomes hypertonic. When water intake is equal to water loss by sweating, theoretically the plasma electrolyte levels, especially that of sodium, should fall. However, owing to the high plasma sodium content and the large extracellular fluid volume, hyponatraemia ([Na^+]<135 mEq/l) is not apparent until late in exercise, after large amounts of water have been ingested with no sodium replacement.

Hyponatraemia is seldom expected in athletes exercising at high intensities for periods of <4 h.[7 11] This conclusion is based on the estimated low total loss of sodium in sweat and the general observation that at high work rates, fluid intake is less than optimal to maintain euhydration. Under these conditions, the prime concern is replenishing body fluid by an adequate drinking routine. During longer periods of exercise, hypovolaemia and dehydration are expected, but hyponatraemia, due to cumulative sodium loss, may also develop; [11] the above examples prove this. It should, however, be noted that slow, unfit athletes are at a higher risk of developing EAH owing to overt overhydration, in the face of their lower sweat rate and relatively large sodium losses in sweat, as recently shown by Almond *et al*[12] during the Boston marathon.

In conclusion, to maintain euvolaemia and serum osmolality, water and sodium intake must compensate for sweat losses. If fluid consumption exceeds sweat rate, dilutional hyponatraemia may occur. As sodium concentration in sweat is low, especially in trained athletes, salt consumption is not required for exercises of short duration (<4 h). However, during prolonged exercise, hyponatraemia will develop even without excess fluid intake, unless sweat sodium loss is adequately replaced. The most effective and physiological method of replacing sodium losses is not necessarily by consuming sports drinks, but rather by appropriate diet. This is the common practice in Israel and the attitude recommended by the American College of Sports Medicine (ACSM).[13] Such a routine prevents dehydration and discourages overhydration, and also prevents the development of hyponatraemia.

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The aetiology of exercise–associated hyponatraemia is established and is not ''mythical''

Epstein and Cohen-Sivan's conclusion that the aetiology of exercise-associated hyponatraemia (EAH) is more complex than our ''oversimplified'' explanation stems from their misunderstanding of how fluid balance is regulated during and after exercise and their ignorance of the conclusions of our most recent publication.[1]

In contrast with their assertion that ''body fluid homeostasis is a primary variable which is important in the stability of the 'internal